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Transfusion-Induced Hypoxemia in Beta-Thalassaemia

To the Editor: Thalassaemic patients frequently present cardiomyopathy; some of them are in a hypoxic state, and they have defective oxygen unloading. In this setting, any additive hypoxemia may lead to deleterious effects. Some authors [1] reported a dramatic drop of PaO_2 (mean 22 mm Hg, range 0–70 mm Hg) following blood transfusion in β-thalassaemia patients. These authors did not mention duration of PaO_2 drop following transfusion; it is also not clear whether such a drop of PaO_2 is confined to thalassaemics.

As our clinical experience on the topic is completely different [2], we reconsidered this issue.

We studied 20 transfusion-dependent β-thalassaemics (age 31 ± 5 years) and 19 patients with anemia of a different etiology matched for age. All thalassaemics were under iron chelation; 12 had been splenectomized. No patient presented with heart failure, whereas 6 were living in a rather hypoxic state. In all subjects, blood was sampled by puncture in a sitting position just before, and 30 min and 24 hr following, transfusion of two blood units. An ABL2 radiometer (Copenhagen, Denmark) was used.

There was no PaO_2 drop at 30-min and 24-hr intervals in all cases studied. Some nonsignificant differences (up to ±8 mm Hg) were within instrumental and individual fluctuations. The relevant PaO_2 mean values (in mm Hg) are depicted in Table I.

According to our findings, it seems that there is no transfusion-related hypoxemia. The findings of Bacalo et al. [1] remain inexplicable, as already mentioned by the authors themselves. However, in any case the implication of microaggregates does not seem plausible for reasons related to the volume of the blood transfused and to the filters in current use. In evaluating the clinical aspects of the procedure, we believe that hypoxia cannot be considered as an obstacle.

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Serum Interleukin-11 in Plasma-Cell Dyscrasias

To the Editor: Intensive research has been devoted to the nature of known and unknown factors able to both stimulate the production of interleukin-6 (IL-6) by the tumor microenvironment and synergize with IL-6 to increase myeloma cell growth. Interleukin-11 (IL-11) is a pleiotropic cytokine that was originally detected in medium conditioned by an interleukin-1α-stimulated primate bone-marrow stromal cell line, PU-34, by its ability to stimulate the proliferation of an IL-6-dependent murine plasmacytoma cell line in the presence of excess neutralizing anti-IL-6 antibodies [1]. IL-11 has also been found to stimulate the T-cell-dependent development of specific immunoglobulin-secreting B cells from murine splenocyte cultures [1] and the differentiation of human B lymphocytes in the presence of accessory cells [2]. In addition, consistent with its in vitro functions, in vivo administration of recombinant human IL-11 to normal mice was found to enhance the generation of immunoglobulin-producing cells [3]. These observations raise interest on the role of IL-11 in plasma-cell dyscrasias, and prompted us to assay this cytokine in the serum of individuals with monoclonal gammopathy of undetermined significance (MGUS) and multiple myeloma (MM).

IL-11 was measured in 48 patients with MGUS, 86 patients with MM in various phases of the disease (46 at diagnosis, 18 in plateau phase, and 22 in progression or relapse), and 33 healthy controls. There were 28 men and 18 women (median age 61 years, range 44–83 years) in the untreated myeloma group. According to the Durie and Salmon staging system, 17 were stage I, 19 were stage II, and 10 were stage III; 6 were substage B. Thirty-six patients were IgG, 6 were IgA, 3 were Bence-Jones myeloma, and 1 was nonsecreting myeloma. Patients in plateau phase were considered to be those with partial or complete response after chemotherapy and a stable disease for at least 6 months. The median age of patients from this group was 62.5 years; at time of diagnosis, 10 were stage II and 8 were stage III. The median age of patients with progressing or relapsed disease was 64 years; 5 were originally stage I, 8 stage II, and 9 stage III. Median time to progression or relapse was 14 months (range, 6–27 months).

Measurements of IL-11 in the sera of patients and controls were carried out by enzyme-linked immunoassay (ELISA) instrumentation with amplified sensitivity (V-MAX Reader, Molecular Devices, Menlo Park, CA) equipped with software for the automatic fitting of standard curves according to preset parameters (the four-parameter logistic equation was utilized). A commercially available kit (Quantikine™ Human IL-11, R&D Systems, Minneapolis, MN) was used according to the manufacturer's instructions. Briefly, this assay was based on a double-antibody sandwich method, had a sensitivity limit of 4 pg/ml, a highest intraassay CV of 4.8%, and a highest interassay CV of 8.0%. As reported by the manufacturer, this ELISA is specific for human IL-11 and does not crossreact with other known cytokines.

Measurable levels of IL-11 were found in 2/33 (6.1%) normals, and in 5/48 (10.4%) patients with MGUS, as compared to 14/86 (17.3%) myelomas (P = NS, χ^2 test). It is noteworthy that in MM the cytokine was detected with a comparable frequency in all pathologic stages and phases of the disease (Fig. 1): 2/17 in stage I, 4/19 in stage II, 2/10 in stage III, 3/18 in plateau phase, and 3/22 in progressing or relapsed disease. Besides, IL-11 levels did not correlate with β_2-microglobulin, C reactive protein levels, or erythrosedimentation rate. When comparing outcome of individuals with detectable and

TABLE I. PaO_2 Mean Values (mmHg) Before and Following Transfusions

Patients studied	Before transfusion	After 30 min	After 24 hr
β-thalassaemia (n = 20)	90.7 ± 10.0	90.1 ± 10.5	89.9 ± 10.1
Other anemias (n = 19)	88.3 ± 9.3	86.7 ± 8.0	89.7 ± 6.5